in other parts of the body. Bendz severely criticizes the interpretation given by Neelsen, of the origin of varices of the portal vein in general. Neelsen has taken no account of the fact that, in all the cases cited by him, the varices occupy the same part of the portal system. Nor can Bendz indorse the theory of Chautemps, referred to by Dussausay. From Zenker's statistics it would seem that true varices of the œsophagus, even in cases of cirrhosis of the liver, are rare.

As regards the anatomy of these varices, the question arises: Does the œsophageal circulation belong to the portal system? Dussauday, relying on the facts in a case examined by him, presumes that most of the œsophageal veins belong to the portal system. Bendz, on the contrary, concludes from the fact that Fürst has examined twelve cases without finding such connections, that it is only in exceptional cases that the œsophagus furnishes a greater or less amount of blood to the portal vein. These exceptional cases, however, are of great importance in the formation of varices of the œsophagus. If, in a parallel case, a trouble of the portal circulation is brought about, there are more open and spacious ways for a supplementary circulation, and varices may be more easily formed than in ordinary cases, in which the communication is made by the submucous veins of the cardia alone. From the situation of the varices in some of these cases, Bendz concludes that they are due to an abnormal distribution of the veins.

The local sequelæ of varices of the œsophagus are dilatation of the canal, causing dysphagia, and erosions of the mucous membrane covering the principal vessels. Their general importance is principally due to the fact that they may cause hemorrhages by rupture or by ulcerations. On the one hand they are of such a nature as to render good service by furnishing a supplementary circulation, and thus preventing ascites. On the other hand, this may render the diagnosis very difficult, especially if the cutaneous veins are not dilated at the same time. From what was found in the first two cases, Bendz does not agree with Dussausay in thinking that the development of varices of the œsophagus, in cases of cirrhosis of the liver, will always prevent ascites.—*Nordiskt Medicinskt Arkiv*, Bd. xvi., Hft. 9.

Infectious and Parasitic Pneumonia.

Prof. GERMAIN SÉE read a paper on this subject before the Académie des Sciences, on Nov. 24th. Whilst of late years, he said, pneumonia has been regarded as the type of simple inflammation, the cause of this inflammation has been attributed to cold. Struck by the observation of a number of cases in which pneumonia behaved as a true infectious disease, successively attacking several members of one family, he has, in his clinical lectures at l'Hôtel-Dieu, opposed the classic doctrine since 1882, and has taught the infectious nature of the disease. Since that time numerous cases, published in England, Germany, and elsewhere, seem to prove that pneumonia may, under certain circumstances, be developed as is typhoid fever, breaking out in a prison, a school, barracks, or a village, and attack a large number of people just as one of the more clearly defined infectious diseases. The question therefore arises: Are these cases to be considered as a special variety of pneumonia or only ordinary pneumonia? The answer is that the identity of the two kinds of pulmonary inflammation has been demonstrated; there is no pneumonia originating from cold; whether occurring as a sporadic or epidemic disease, pneumonia is always parasitic.

The micro-organism of pneumonia was simultaneously demonstrated in Germany by Friedländer, and in France by Talamon, Prof. Sée's Chef de Clinique. According to Talamon, the parasite of pneumonia is an oval micrococcus, from 1 μ to 1.50 μ long, and from .50 to 1 μ broad; it is constantly found in hepatized lung tissue. As seen in the fibrinous exudation, it has the form of a grain of wheat. Cultivated in a liquid medium, in alkalinized solution of meat extract, it is elongated, tapering, and looks like a grain of barley. It is sometimes single, sometimes coupled two together as diplococci, and sometimes occurs in chains of four. Friedländer's description is very similar to that of Talamon, but he claims to have also found, besides the pneumonic cocci, a capsule which he regards as the essentially characteristic element of the micrococcus. This capsule does not exist, and has nothing to do with the micrococcus: it is a result of his method of preparing and coloring the exudation. Fränkel, at the last Berlin Congress, declared that the capsule is not a constant phenomenon; that it may be found entirely irrespective of any micro-organisms, and that the capsules cannot possibly be considered as characteristic of the micrococci of pneumonia; and Friedländer has recently renounced the idea that it is in any way distinctive of the pneumonic micro-organism.

Whatever it may be, this micrococcus is not found in the inflamed lung alone; it has been isolated and cultivated in a liquid and solid medium; and, inoculated in animals, has produced lobar pneumonia, such as is seen in man. Thus far, no irritating substance introduced into the respiratory passages has caused lesions characteristic of frank pneumonia; a special action of the micro-organism is needed to cause it. Friedländer and Talamon, by injecting cultures of the micrococcus into the lungs of mice and rabbits, have caused typical cases of pneumonia, precisely similar to that occurring in man. In a number of cases the microbe, by emigrating from the lung and invading neighboring organs, has caused at the same time pleurisy and fibrinous pericarditis of the same nature as the pulmonary inflammation.

From these facts we cannot hesitate to regard pneumonia as a specific parasitic disease; it may be reproduced in animals, but the reproduction is impossible with ordinary irritants, physical or chemical. In order that the characteristic inflammation be developed, it is necessary that a special microphyte be brought in contact with the lung tissue, and there multiply. The inflammation remains localized as long as the micro-organism does not pass the limits of the pulmonary tissue; this is *simple* pneumonia. It extends and becomes generalized when the microbe invades the general circulation, either through the lymphatic or vascular system; when it becomes *infectious* pneumonia.

This parasitic pneumonia may be absolutely distinguished from the other forms of acute pulmonary inflammation, usually described under the name broncho-pneumonia. Without doubt, microbes also play a part in these cases; but that part is secondary; the course of the disease, as well as the extension of the lesion, is here dominated by the existence of a previous bronchitis, due to the influence of cold, and by the propagation of this bronchitis to the finer bronchi and the alveoli. Broncho-pneumonia, developed from bronchitis *a frigore*, has nothing definite in its evolution; it proceeds by successive and irregular stages. Like the eruptive fevers, parasitic pneumonia has a definite course. Its duration is within fixed limits, and usually does not last more than six or nine days; it is characterized by intense hyperpyrexia; the temperature keeps at one point for about a week, following an unvarying course, as in all acute infectious diseases, dominating during this period all local manifestations and physical signs, and then falls suddenly, often on the seventh day.

As regards treatment, our object should be to tide the patient over the seventh day, as the disease usually declines at this time. Venesection and tartrate of antimony should not be used. The temperature should be kept down by quinine and antipyrine; the patient kept up by alcohol and nourishing liquid food.—*L'Union Médicale*, Nov. 29, 1884.

Sclerosis of the Coronary Arteries and the Affections depending upon it.

LEYDEN, in an article on this subject in the *Zeitschrift für klinische Medicin*, Bd. vii., says that since a deeper insight has been made into the relations of certain pathological changes of the heart with diseases of its vessels, the attention of clinicians has been drawn to sclerosis of the coronary arteries, and the affections which seem to depend upon it. Samuelson found that confinement of or pressure upon the coronary arteries caused a marked diminution of the cardiac contractions, with corresponding lowering of the blood-pressure; whilst Cohnheim and Schulthess-Rechberg observed that after a previous state of irregularity of the pulse, the strongly-beating heart suddenly stops.

Leyden gives the following as the pathological anatomy of the affection:—

1. Sclerosis or ossification of the coronary arteries, without special changes in the heart. The heart-muscle does not suffer, and the patient dies of an intercurrent disease.

2. Acute thrombotic softening, or formation of hemorrhagic infarctions in the heart-substance, consequent upon sclerotic thrombosis of the diseased arteries—myomalacia cordis of Ziegler. This leads, at a later period, either to fatty degeneration in the occluded vascular districts, or to softening, the most frequent cause of rupture of the heart.

3. The chronic form, fibrous degeneration of the heart-muscle, myocarditis fibrosa, arising from the callous-formations in the affected vascular districts. In this manner, also, aneurism of the heart is frequently produced at the apex.

4. A combination of the above-mentioned varieties, the most frequent of all. Clinically, these forms are seen in a more or less acute, or more chronic form, and Leyden classifies them as—

(1) Acute cases, with sudden termination in death. Death occurs in a fainting fit, or after an attack of angina pectoris. In some cases an autopsy shows very marked, in others very slight, anatomical changes in the heart: rupture of the heart, hemorrhagic infiltration with extravasation of blood into the pericardium, softening or only small foci of fatty degeneration or slight softening of the heart-muscle. Whilst these cases are reported as cases of embolism of the coronary arteries, we have the symptom of slowing of the pulse (Samuelson) frequently very pronounced, or sudden paralysis of the heart.

(2) Cases of subacute course, showing that the severe course of the disease runs through a number of weeks. There are certain forerunners of the disease—as cough, dyspnœa, frequent attacks of angina pectoris, sometimes even dropsy. The patient may then improve for a while, when severe symptoms will suddenly appear, and death take place. An autopsy will show myomalacia, hemorrhagic infiltration, fatty degeneration, and signs of old fibrosis.

(3) Chronic cases, generally described as chronic myocarditis, fibrous myocarditis, dilation or aneurism of the heart. In persons of advanced age, heart-symptoms develop slowly, and sometimes disappear. There may be attacks of angina pectoris or cardiac asthma. The course of the disease is almost always progressive; whilst the symptoms increase the patient presents the phenomena of severe disease of the heart, and under the increase of the asthmatic attacks, and the appearance of dropsy—and not very seldom of delirium, sometimes, also, in sudden attacks—the disease terminates fatally.

The symptomatology of sclerosis of the coronary arteries is that of an acute or chronic disease, with more or less striking phenomena, independent of valvular affections of a progressive character, coming at an advanced age, and leading to a diminished functional activity of the heart. The cardiac symptoms are prominent, the vital or physiological more so than the physical; and angina pectoris, asthmatic attacks, weakness of the heart, fainting fits, changes in the heart's activity, both in frequency and rhythm, and in some cases a prolongation of the diastolic period, are seen in these cases. Physical examination shows dilation of the left ventricle, whilst the right is usually hypertrophied. The complications on the part of the respiratory apparatus are cough, dyspnœa, catarrhal affections and attacks of pulmonary œdema. The most frequent causes of arterial sclerosis are heredity, the abuse of alcohol, and high living. Physical and mental strains also play an important part.—*Centralbl. für klin. Medicin*, Sept. 20, 1884.

Acute Pancreatitis in Child-bed.

Dr. R. HAIDLEN, of Stuttgart, reports the following case from Dr. Fehling's private practice:—

A woman, æt. 33 years, became pregnant, and during the period of gestation had considerable gastric disturbances and headache. She was delivered with the forceps, there was no post-partum hemorrhage, and the after-period was normal, until the third week after delivery, when there was some hemorrhage. A little later she had two attacks of pain in the region of the stomach, and five and a half weeks after delivery she had a very severe attack of pain in the pyloric region, accompanied by vomiting. There was no elevation of temperature, the pulse was 100–104, and regular. The epigastric region was sensitive to pressure, but there were no symptoms of peritonitis; the skin was pale, and not icteric. The patient seemed somewhat collapsed. In the afternoon of that day she seemed worse, the skin was paler, the pulse small and more frequent (110 to 120), and the vomiting had ceased. The sensibilities were normal.

Physical examination showed marked swelling of the abdomen, great sensibility of the pyloric region, the parametrium free, the uterus small, movable, not sensitive; there was diarrhœa; the urine was passed spontaneously in considerable quantity; there was a considerable amount of gas in the intestines. Warm fomentations were placed over the painful regions, subcutaneous injections of camphor were ordered, and champagne and fluid diet. On the following day the patient was better, but the symptoms returned, and she died collapsed in ninety-six hours.

The autopsy showed that there was no peritonitis, though a small amount of a dirty bloody-looking fluid in the lower part of the peritoneal cavity. The organs, with the exception of the pancreas, were normal. Examination of the pancreas showed that it had undergone considerable changes; it was longer, thicker, and broader than normal, and in only a few places was its normal color retained; it had changed almost entirely to a brownish-red, blood-suffused mass, containing a small clot of blood on the anterior surface, in contact with the mesentery, but there was no perforation anywhere. The adjacent portion of the mesentery was suffused with blood.

The microscopic examination was made by Prof. Ziegler. He made the diagnosis as acute pancreatitis, with hemorrhage into the tissue of the organ; the microscope also showed small-celled infiltration of the pancreatic tissue. The duct of Wirsung was somewhat dilated.

Dr. Haidlen, after an extensive search for the report of a similar case, has found an account of the symptomatology of acute pancreatitis only in Strümpel's *Lehrbuch der Speciellen Pathologie und Therapie*, Bd. i. p. 714. A few cases of inflammation of the pancreas are found in literature; the affection commences with severe colicky pain in the epigastrium, and vomiting and collapse soon follow. The pulse is small, the extremities cool, and death occurs in a short time. The pathological appearances are such as have already been described. As to the etiology, nothing is known.—*Centralbl. für Gynäkologie*, Sept. 27, 1884.